specimen tested by different urinometers, and by another physician as well as myself, was 1004. The patient had been drinking beer with some friends all the morning, an explanation of the intoxication, the polyuria, and the low specific gravity.

On looking up the subject I find that even lower specific gravities than this are reported as occasionally met with. Of 224 cases in one of Külz's series, twenty-seven, or 12 per cent., showed at times a specific gravity of less than 1010. In one case it was as low as 1003. Eichhorst says that once in a while a specific gravity as low as 1008 or 1002 is seen. The latter case is perhaps the one Waterman reported in the *New York Medical Record* in 1882. Naunyn found a specific gravity of 1003 in spite of over 1 per cent. of sugar. And many other cases with occasional specific gravities below 1010 are reported. The lesson is, therefore, plain that the only safe way is to examine every urine for sugar, even when the specific gravity is low. One patient may be in the incipient stage of a complicating chronic nephritis, may be weak and debilitated, or may have been consuming large amounts of liquids, any one of which circumstances may tend to lower the specific gravity of the sugar-containing urine. And the importance of this fact to the life insurance examiner needs no elucidation. That some companies to-day only require the examiner to test for sugar when the specific gravity is above 1020 or perhaps 1015 shows that the occasional occurrence of saccharine urine of low specific gravity is not as generally recognized as it should be.

PNEUMONIC COMPLICATIONS IN PULMONARY PHTHISIS.[1]

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Ever since the lungs of phthisical patients have been examined by pathologists the variety of lesions observed has made a great impression on all careful investigators. The variety is such that almost by itself the question arises whether all these different lesions could really be produced by one and the same disease, or whether several diseases working together were at the bottom of them.

This question found different answers by different observers, and a very heated scientific discussion arose over the question of the " unity " or " dualism " in the phthisical process. The most important exponent of the doctrine of the unity of tuberculosis was the great French clinician and anatomist, Laennec, whereas Virchow differentiated strictly between the tuberculum proper and the more diffuse caseous pneumonia.

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[1] Paper read at the thirtieth annual meeting of the Medical Society of the State of California, at San Francisco, April 17, 1900.

The question seemed to find a solution when, in 1882, Koch discovered that in all tuberculous lesions it was possible to find the same one organism, the tubercle bacillus, but Koch himself doubted whether this one organism alone could produce the great variety of lesions which we find in the phthisical lungs, and suggested that in its destructive work in that organ it might be frequently assisted by other pathogenic bacteria; and a number of his pupils, among whom I should like to mention Spengler especially, have endeavored to ascertain how far in the production of the pulmonary lesions the tubercle bacilli themselves were concerned, and how far they were the result of a mixed infection with the tubercle bacilli and other pathogenic bacteria. In 1891, Orth made a special study of the pneumonic complications in pulmonary phthisis, and he came to the conclusion that the tubercle bacillus could produce in a general way, but especially in the lungs, two forms of lesions—one of them of nodular appearance, the tuberculum proper; the other a more diffuse pneumonic process with consecutive caseation, the so-called caseous pneumonia. The question how far in the production of these caseous pneumonias mixed infection was concerned he left in doubt, but he expressed his belief that the tubercle bacillus alone could produce at least certain forms of them, because in a number of cases he had searched for other bacteria and found none. The question was then taken up in 1893 by Ortner, who came to the opposite result, and claimed that the pneumonic complications of phthisis were due in all instances to mixed infection, although in a few of his cases he himself was not able to demonstrate any other bacteria besides the tubercle bacilli; but soon afterward, in 1894, Fränkel and Troje were able to prove that such an extreme view was not supported by the facts. They demonstrated conclusively that there are at least certain forms of caseous pneumonia in which at the post-mortem we do not find any other bacteria but the tubercle bacilli, and which, therefore, appear to be the result of a simple, not of a mixed infection. But these authors in their turn seem to have gone to an extreme in giving too little importance altogether to the mixed infections which undoubtedly occur quite often. In 1895, Professor Orth, in Göttingen, suggested to me to make further investigations in this direction, and since that time I have been interested in this important question. Part of the material used for the present paper was collected in Göttingen, and I desire to express my sincerest gratitude to Professor Orth for allowing me to make use of the same, and also for many valuable hints in regard to the method of investigation pursued; the other part of the material has been collected since I came to San Francisco.

In this necessarily short introduction I have mentioned only a few of the most important papers, and could therefore not do justice to a host of careful investigators who have treated the subject from one stand-point or another; but I hope to give a more detailed historical review in a more extensive paper on the same subject to be published later.

When we try to approach our subject systematically the first question to be answered would be, Where does the infectious material that causes the pneumonic complications come from? There seems to be hardly any doubt that in most instances this infectious matter is furnished by the pulmonary cavities, at least all observers agree that in the vast majority of cases in which we find pneumonic complications we also find cavities containing such matter as would be apt to produce them when carried into healthy portions of the pulmonary tissue. Since the larger cavities are almost invariably in direct communication with the bronchial tubes, and thus with the air, they usually do not contain tubercle bacilli in pure culture, but other micro-organisms besides, sometimes saprophytes, but mostly pathogenic forms. Of the latter the following have been met with: staphylococcus pyogenes aureus and albus, streptococcus pyogenes, pneumococcus, micrococcus tetragenus, Friedländer's pneumobacillus, influenza bacillus, colon bacilli, diphtheria and pseudodiphtheria bacilli.

I myself have examined twenty-six cavities in thirteen cases. I found a pure culture of tubercle bacilli in seven of these twenty-six cavities; the others contained a mixture of tubercle bacilli and other bacteria. The species encountered were the following: streptococcus pyogenes in one case; pseudo-diphtheria bacilli in five cases; pneumococci in six cases; pneumococci and pseudo-diphtheria bacilli in four cases; staphylocccci and streptococci in two cases; pneumococci and staphylococci in one case.

The infectious material is carried from the cavities to other parts of the lung by what is commonly called aspiration. The process in reality often is not so much of an aspiration as a gravitating downward of the infectious material in the bronchial tubes. Whether a pneumonic process is set up by such an aspiration depends largely on the number, the variety, and the virulence of the bacteria present in the material aspirated.

The point to which the infectious matter is carried also plays an important rôle. Pneumonic complications will naturally arise only when the material is carried at least as far as the infundibula.

In those cases in which the cavities are not surrounded by a continuous membrane of granulation and scar tissue the pathogenic organisms may enter adjoining healthy portions of the lung directly from the cavities, without travelling first through the bronchial tubes.

In all I have examined the lungs of fifty-six phthisical patients with regard to pneumonic complications, and among thirty-seven successive cases I have been able to find pneumonic complications in twenty-five instances. Of course, it is entirely impossible to examine every part of a phthisical lung minutely, so I am ready to acknowledge that in some cases smaller pneumonic patches may have escaped my attention. The lesions that I have found have naturally not been of the same character in different parts of the same lung, and so the number of specimens actually examined has been much larger than the number of cases. The results of my examinations have been, shortly, the following:

In the first place I am able to confirm the statement made first by Orth, and later corroborated by Fränkel and Troje, namely, that there is one form of pneumonic process met with in tuberculous lungs which seems to be produced by a simple infection with tubercle bacilli. At least when we examine these pneumonic patches after the death of the individual we do not encounter any other bacteria in them. What makes it even more probable that they are the results of a simple infection with tubercle bacilli is the fact that these lesions exhibit histological characteristics by which they differ considerably from that form of bronchopneumonia which arises from a mixed infection. We can distinguish between different forms of this true tubercular pneumonia. The first one we might call the acute type. In it we find a central caseous area surrounded by air spaces filled with fibrin, epithelioid cells, and lymphocytes. The more peripheral air spaces usually contain desquamated epithelium, a few lymphocytes, œdema, or a little fibrin. The septa do not seem to participate actively in the process at all. The capillaries are often hyperæmic; occasionally there are hemorrhages. Of such acute cases I have seen eleven examples, of which nine contained tubercle bacilli in pure culture; in the other two, cultures made from the affected spots contained a few pus cocci; but in spite of careful search for them they could not be found in sections, and were therefore most likely derived not from the diseased area itself, but accidentally gathered from cut small bronchial tubes. In the more chronic cases we find between the exudate and the caseous area a layer of tuberculous granulation tissue which pushes its way more or less far into the adjoining air spaces. The granulation tissue sometimes contains giant cells. The septa are either normal or show a moderate degree of infiltration with lymphocytes, and only rarely are they appreciatively thickened by the development of granulation tissue in them. Such more chronic tuberculous lesions were found eighteen times, and in all cases they did not contain any other bacteria but tubercle bacilli. The most chronic forms are those in which the tuberculous granulation tissue has a tendency to become transformed into a simple granulation tissue, in which, therefore, the tubercular process becomes a simple carnification —in other words, shows a tendency to heal. This I have seen twice.

Between these three forms of true tubercular pneumonia we naturally find all transitional stages, since essentially the process is one and the same, and differs only quantitatively, so that in a given specimen it may be difficult to classify the lesion among one of these three groups.

The acuteness of the process seems to depend to a certain extent on the number of tubercle bacilli present in the diseased spot, so that in the cases with many tubercle bacilli the lesions had an acute character. But this correspondence between the acuteness of the lesion and the number of tubercle bacilli is not complete. The virulence of the bacilli and the condition of the patient therefore also seem to exercise an important influence upon the amount and character of the reaction in the tissues.

Here I should like to mention shortly an objection that has been raised by Baumgarten against Orth, namely, whether the processes just described were really rightly called tuberculous *pneumonia*. Baumgarten, and with him Troje, claims that inasmuch as in them we observe the appearance of epithelioid cells in the air spaces and in the more chronic forms, even of a more or less typical tuberculous granulation tissue, they do not differ materially in their structure from ordinary tubercles, as we find them in miliary tuberculosis of the lungs or other organs, the only point of difference being that in these cases the tubercular tissue develops inside of the air spaces. But Baumgarten and his followers seem to overlook entirely that with the presence of the epithelioid cells, etc., we have in the tubercular pneumonias an inflammatory exudation which is best shown by the constant presence of a very large amount of fibrin in the lesions—a fact which was first duly emphasized by Orth. It is true that fibrin has been found in miliary tubercles also (Falk), but only in a very small quantity. Now, it seems that it is a general principle in pathology that " a potiori fit denominatio." These lesions, therefore, are rightly classified among the pneumonic processes, whether the exudate contains epithelioid cells or not; otherwise one might just as well object to calling a chronic abscess an abscess because the pus in it contains a certain number of fibroblasts.

Such an area of tuberculous pneumonia may become the seat of a secondary infection with other pathogenic bacteria by way of the bronchial tubes; and then the bacteria, mostly pus cocci, seem to find a soil very suitable for their development in the caseated matter, and in developing in it they seem to add very considerably toward its rapid disintegration. This corresponds well with the results of some animal experiments made by Prudden, who was able to produce cavities in the lungs of tuberculous animals by injecting pure cultures of pus cocci into their bronchial tubes, causing in that way a secondary infection of the tuberculous areas in them.

But although secondary infections favor the rapid disintegration of the caseous material, and with that the formation of cavities, they are by no means necessary to this end. I have frequently seen evidence of a breaking down of caseous material without secondary infection. In these latter cases I have never missed finding a very large number of tubercle bacilli in the disintegrating material.

The extent of the lesion, whether it affects only small portions of a lobe or whether it involves an entire lobe, or even an entire lung, depends, of course, entirely on the amount of material aspirated and the way it is accidentally distributed in the pulmonary tissue.

There remains one question of great clinical interest, namely, What is the ultimate outcome of these tuberculous pneumonias? In most cases the local outcome seems very unfavorable in that they lead to rapid caseation and disintegration of the pulmonary tissue. The rapidity of the process varies, of course, with the more acute or more chronic character of the process, but the few specimens in which we find little caseation and a transition of tuberculous into simple granulation tissue seem to prove that sometimes even these forms of tuberculosis may exhibit marked tendency toward healing.

In other forms of pneumonic complications we have mixed infections from the beginning. In these cases other pathogenic bacteria are carried simultaneously with the tubercle bacilli into the healthy parts of the lungs. Here also we can distinguish between more acute and more chronic forms. The acute forms present to the naked eye entirely the aspect of ordinary bronchopneumonias, and do not differ materially from them in their microscopical structure. In these first stages of the process caseation may not be present. In some cases they betray their at least partly tubercular nature by the presence of epithelioid cells in the exudate. In the more chronic cases of mixed infections we find either a very marked tendency to rapid caseation or disintegration, or, on the contrary, the process may also end in carnification.

Among sixteen cases of mixed infection there were nine cases of acute bronchopneumonia without caseation; four cases with apparently very rapid and extensive caseation, and four cases with carnification. The bacteria, found together with the tubercle bacilli, were the following: Pneumococci, ten cases; streptococci, one case; pneumococci and streptococci, one case; pseudodiphtheria bacilli, one case; pseudodiphtheria bacilli and streptococci, one case; cocci of unknown nature, in one case.

In one case, which histologically had all the characteristics of an acute mixed infection, I was unable to find any bacteria besides a few tubercle bacilli. The only explanation that I can offer in this case is the supposition that other bacteria had been present, but were already overcome and destroyed at the time of the post-mortem.

It is interesting to note that the only two cases in which there was an infection with pseudodiphtheria bacilli there also was carnification.

Besides these two outcomes caseation on the one hand and carnification on the other, there seems to be one other of which it is not possible to obtain anatomical proof. It is very likely, and seems to be supported by clinical evidence also, that not rarely the acute pneumonic process set up by a mixed infection heals. Clinically the recovery may be and often is complete, but the presence of tubercle bacilli in these lesions makes it very probable that anatomically the process will not heal completely, but with a remaining more or less extensive simple tubercular lesion.

Ordinary lobular and lobar pneumonias naturally may occur in the course of phthisis, but they do not seem to be frequent. At least among my cases I have been able to find only two cases of bronchopneumonia and one case of lobar pneumonia in which no tubercle bacilli could be discovered in the exudate. It has often been asserted that such simple pneumonic complications could be secondarily infected with tubercle bacilli, but as far as I see it has not been possible to furnish an anatomical proof for such an assertion.

There is also the possibility that acute mixed infections may in course of time assume the character of a simple tubercular pneumonia after all other bacteria except the tubercle bacilli have been destroyed in them. In view of the case in which the lesions showed the histological characteristics of a mixed infection, and nevertheless did not contain any other bacteria but tubercle bacilli, I think that such may be the case; and when we consider that cavities rarely contain a pure culture of tubercle bacilli, whereas simple tubercular pneumonias are met with quite often, we might argue that such an occurrence is perhaps more frequent than we are inclined to believe, when we judge merely from the anatomical and bacteriological findings at the post-mortem.

When we try to get a correct idea of the general importance of the pneumonic complications in phthisis, all forms included, it becomes of importance to note how frequently they are met with, and, from an anatomical stand-point especially, how frequently they are the immediate cause of death. As I have mentioned above, I have found pneumonic complications among thirty-seven successive cases twenty-five times, showing that, at least in the later stages of the disease, they occur most frequently, and I am sure that in most of these cases the pneumonic complications were the direct cause of death. Frequently they seem to be accompanied with acute generalized infections with the bacteria that are associated with the tubercle bacilli in causing them. At least among seven cases of acute mixed infections in which I have records of a bacteriological examination of other organs, there are five with more or less complete generalization of the infection. In one of them there existed a general infection with the pneumococci, in another a general infection with Friedländer's bacilli. In two of the cases pneumococci were found in the lungs and liver, and in one pseudodiphtheria bacilli also in lungs and liver.

1. True dermoids.
2. Dermoids containing cartilage and cylindrical cells.
3. Dermoids combined with lymphoid tumor.

That a division into these three groups is not a satisfactory classification will presently be shown in discussing the embryology of the subject. In the case just described the cyst was for a time considered to belong to Hoffmann's first group. After making sections from various parts of the cyst walls the presence of cartilage was finally demonstrated. Several authors have reported cases of dermoid cyst in the thoracic cavity, but have failed to mention them as originating in the mediastinum. It is the writer's opinion that in most instances the mediastinum will be found to be the original source of the cyst.

Pflanz[4] has made a more careful search of the literature and has collected a series of twenty-four cases, to which he has added a personal case. The patient was a man, aged twenty-one years, who presented a cyst in the anterior mediastinum below the right clavicle and bulging forward behind the first and second ribs on the right side. The cyst was opened by an operation and drained. It contained hair, fat, and dermoid detritus, and the patient left the hospital with a narrow fistula remaining. The reader is referred to the original article for a careful description of the twenty-four recorded cases.

Ekehorn[5] has reviewed the cases tabulated by Pflanz, and has added to the list five additional cases from the literature and two personal cases. His first case was that of a woman, aged twenty-one years, with a cyst in the lower portion of the right chest attached to the mediastinum. Bone and cartilage were seen, and between some of the small areas of cartilage ganglion cells and nerve fibres were present; ciliated epithelium was also found. The largest single piece of bone was 1½ cm. x 2 cm. in size, from which several teeth protruded. Its whole appearance was similar to a portion of the upper jaw. The second case was a patient of Dr. Fogman's, in Stockholm, who presented a cyst to the left of the heart and attached to the left lung. Two teeth protruded from a polypoid growth in the cyst cavity. The author did not have access to the account of Smythe's case, which he mentions only by title, so I shall insert it here.

Smythe[6] describes the case of an unknown woman, aged thirty years, who died suddenly. The autopsy revealed the presence of a cyst occupying the entire upper lobe of the left lung, and pressing upon the pericardium and mediastinum. The cyst contained dermoid tissue and hair, and in its cavity was found a bony mass weighing eight grammes. The report does not state whether the cyst was a true mediastinal one, but it undoubtedly belongs to this class.

Another case not mentioned by either Pflanz or Ekehorn is that published in 1893 by Kretz.[7] The patient was a man, aged thirty years, in whom the diagnosis of pulmonary tuberculosis and bronchiectasis in at essentially the same conclusions. There are only a few points in which my observations are at variance with those of Professor Sata. In the first place, he does not seem to have found pure cultures of tubercle bacilli so frequently in open cavities as I have. When he finds in his specimens almost exclusively what he calls "mischpneumonie," that is, lesions partly due to tubercle bacilli, partly to infection with other bacteria, whereas, according to my statements, it is not so unusual to find pneumonic lesions in which we find tubercle bacilli only. This apparent discrepancy may be due largely to his not having separated so strictly as I have the results of mixed infection and those of apparently simple infection with tubercle bacilli which often occur in immediate proximity to one another in one and the same specimen. However that may be, we both agree in regard to the main point—*i. e.*, the importance of mixed and secondary infections in the development of the destructive process in phthisical lungs.

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A CASE OF DERMOID CYST OF THE MEDIASTINUM,

WITH REMARKS UPON THE ETIOLOGY AND EMBRYOLOGY, AND A SURVEY OF RECENT CASES.

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A brief pathological account of the following case was published in the *Mount Sinai Hospital Reports*, 1898, vol. i., but a minute description has purposely been delayed in the expectation that another instance of this rare disease might present itself. In this report no cognizance has been taken of differential diagnosis, nor has a detailed account been given of the cases hitherto tabulated by previous writers on the subject. The history of the case follows:

Fanny G., aged thirty years, a native of Russia, was admitted to the hospital on January 24, 1898. Her family history was entirely negative.